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| EXAMINER | |
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| SITTON, JEHANNE SOUAYA | |
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**Please find below and/or attached an Office communication concerning this application or proceeding.**

The time period for reply, if any, is set in the attached communication.

| Office Action Summary | Application No. | Applicant(s) | |
|---|---|---|---|
| | 10/073,123 | LI ET AL. | |
| | Examiner | Art Unit | |
| | Jehanne S. Sitton | 1634 | |

*-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --*

**Period for Reply**

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

**Status**

1)☒ Responsive to communication(s) filed on *29 April 2008*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

**Disposition of Claims**

4)☒ Claim(s) *1,3,57-59 and 61-63* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1, 3, 57-59 and 61-63* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

**Application Papers**

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

**Priority under 35 U.S.C. § 119**

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

**Attachment(s)**

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

## DETAILED ACTION

1. Currently, claims 1, 3, 57-59 and 61-63 are pending and under consideration in the instant application. All the amendments and arguments have been thoroughly reviewed but are deemed insufficient to place this application in condition for allowance. The following rejections are reiterated. They constitute the complete set being presently applied to the instant Application. Response to Applicant's arguments follow. This action is FINAL.

2. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

3. The rejection of claims 54 and 56, made under 35 USC 112/second paragraph is moot in view of the cancellation of the claims.

### *Maintained Rejections*

### *Claim Rejections - 35 USC § 112*

### *Written Description*

4. Claims 1, 3, 57-59 and 60-63 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The claims recite "detecting and measuring gene copy number of a WIP1 gene in a breast tissue…". The designation of WIP1, however, includes a large genus of molecules, including mutants, variant, and homologs of SEQ ID NO: 1 and SEQ ID NO: 3, as exemplified by the definition in the specification. The specification defines "WIP1" as nucleic acid which can

include their polymorphic variants, alleles, mutants that have substantial nucleotide sequence homology with the nucleotide sequence of GenBank entry AAB61637 or "substantial" nucleotide sequence homology with the nucleotide sequence as set forth in SEQ ID NO 1 (para bridging pages 21 and 22 of the specification). Although the specification does not provide an express definition for substantial nucleotide sequence homology in the context of nucleic acids, at page 21, the specification provides a broad definition with regard to protein homology as having only 60% identity. Therefore, it is clear that the term "substantial" allows for a large degree of variability when comparing two sequences. Accordingly, it is clear from the guidance in the specification that the term "WIP1" is not limited to the gene which encodes the SEQ ID NO: 1 transcript, but also broadly encompass mutants, allelic variants, and homologs. However, the specification only teaches the sequence of SEQ ID NO: 1 and 3, which correspond to WIP 1 coding sequence. Neither sequence is the sequence of the WIP1 gene, nor is it representative of the broad genus of mutants, variants, and especially homologs of WIP 1 with altered structure or function, as well as occurring at a different region of the human chromosome, let alone chromosome 17. Based on the broad definition of the WIP1 gene (nucleic acid sequences that have substantial nucleotide sequence homology with the nucleotide sequence of the GenBank entry AAB61637 or SEQ ID NO:l), the large genus of WIP1 genes recited in the instant claims encompasses structurally and functionally distinct molecules, which have not been taught or described in the specification, whose amplification would not necessarily be expected to be associated with breast cancer.

***Response to Arguments***

5. The response does not address this rejection.

***Enablement***

6. Claims 1, 3, 57-59 and 61-63 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of diagnosing breast cancer in a human comprising detecting and measuring gene copy number of the WIP1 gene which encodes the transcript of SEQ ID NO: 1 in a breast tissue sample from the human that is suspected to be cancerous, thereby generating data for a test gene copy number and comparing the test gene copy number to data for a control gene copy number, wherein amplification of the gene in the breast tissue sample relative to the control indicates the presence of breast cancer in the human, does not reasonably provide enablement for a method a method of diagnosing breast cancer in a human comprising detecting and measuring gene copy number of any "WIP1" gene in a breast tissue sample from the human that is suspected to be cancerous, thereby generating data for a test gene copy number and comparing the test gene copy number to data for a control gene copy number, wherein amplification of the gene in the breast tissue sample relative to the control indicates the presence of breast cancer in the human. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make or use the invention commensurate in scope with these claims. There are many factors to be considered when determining whether there is sufficient evidence to support determination that a disclosure does not satisfy the enablement requirements and whether any necessary experimentation is undue. These factors have been described by the court in *In re Wands*, 8 USPQ2d 1400 (CA FC 1988). *Wands* states at page 1404,

> "Factors to be considered in determining whether a disclosure would require undue experimentation have been summarized by the board in Ex parte Forman. They include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence or absence of working examples,

(4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims."

The nature of the invention and the breadth of the claims:

The claims are drawn to a method of diagnosing breast cancer in a human comprising detecting and measuring gene copy number of any "WIP1" gene in a breast tissue sample from the human that is suspected to be cancerous, thereby generating data for a test gene copy number and comparing the test gene copy number to data for a WIP1 control gene copy number and identifying the breast tissue samples as cancerous if there is amplification of the WIP1 gene in the breast tissue sample.

The invention is in a class of inventions which the CAFC has characterized as 'the unpredictable arts such as chemistry and biology" (Mycolgen Plant Sci., Inc. v. Monsanto Co., 243 F.3d 1316, 1330 (Federal Circuit 2001)).

The amount of direction or guidance:

The claims recite "detecting and measuring gene copy number of a WIP1 gene in a breast tissue…". The specification teaches that WIP1 is a serine/threonine specific protein phosphatase type 2C (PP2C) family member whose expression is induced in response to gamma or UV radiation in a p53-dependent manner (pages 37 and 38). The designation of WIP1, however, includes a large genus of molecules, including mutants, variant, and homologs of SEQ ID NO: 1 and SEQ ID NO: 3, as exemplified by the definition in the specification. The specification defines "WIP1" as nucleic acid which can include their polymorphic variants, alleles, mutants that have substantial nucleotide sequence homology with the nucleotide sequence of GenBank

entry AAB61637 or "substantial" nucleotide sequence homology with the nucleotide sequence as set forth in SEQ ID NO 1 (para bridging pages 21 and 22 of the specification). Although the specification does not provide an express definition for substantial nucleotide sequence homology in the context of nucleic acids, at page 21, the specification provides a broad definition with regard to protein homology as having only 60% identity. Therefore, it is clear that the term "substantial" allows for a large degree of variability when comparing two sequences. Accordingly, it is clear from the guidance in the specification that the term "WIP1" is not limited to the gene which encodes the SEQ ID NO: 1 transcript, but also broadly encompass mutants, allelic variants, and homologs. However, the specification only teaches the sequence of SEQ ID NO: 1 and 3, which correspond to WIP 1 coding sequence. Neither sequence is the sequence of the WIP1 gene, nor is it representative of the broad genus of mutants, variants, and especially homologs of WIP 1 with altered structure or function, as well as occurring at a different region of the human chromosome, let alone chromosome 17. Based on the broad definition of the WIP1 gene (nucleic acid sequences that have substantial nucleotide sequence homology with the nucleotide sequence of the GenBank entry AAB61637 or SEQ ID NO:1), the large genus of WIP1 genes recited in the instant claims encompasses structurally and functionally distinct molecules, which have not been taught or described in the specification, whose amplification would not necessarily be expected to be associated with breast cancer.

Presence and absence of working examples:

The specification teaches that the WIP1 gene is amplified and/or overexpressed in several breast tumor cell lines (Table 1). The specification also teaches that the WIP1 gene is

overexpressed in several primary tumor samples of different types of cancer and amplified in several primary breast tumor samples (Table 2). The specification further teaches methods for detecting and quantitating WIP1 gene amplification and level of expression (pages 40-45). At page 64, the specification teaches a working example of detecting WIP1 amplification using microarray based CGH (comparative genomic hybridization). The specification teaches using a TaqMan probe set representing the target and a reference probe representing normal non amplified, single copy region in the genome. At page 66, the specification teaches that the inventors demonstrated that WIP1 is located at the epicenter of the amplification region using Q-PCR and fluorogenic TaqMan probes based on undisclosed EST's or BAC sequences. The specification, however, does not teach any working examples of diagnosing breast cancer in humans by detecting amplification of any WIP1 gene homologs, as is broadly encompassed by the claims.

The state of the prior art and the predictability or unpredictability of the art:

With regard to an association of the amplification of homolog variants of the WIP1 gene with cancer, Lavi et al. teach that PP2Calpha was expressed at lower levels in 7 out of 8 colorectal tumors compared to adjacent normal colon tissues, suggesting that amplification of the PP2Calpha homolog of the WIP1 gene is not associated with cancer (see Lavi et al., WO97/10796, page 46, lines 19-22).

The level of skill in the art:

The level of skill in the art is deemed to be high.

The quantity of experimentation necessary:

Based on the lack of guidance in the specification and the unpredictability in the art, it would require undue experimentation for the skilled artisan to practice the invention as broadly as it is claimed. The skilled artisan would be required to test a large number of patients and controls to determine whether an association existed between amplification of WIP1 homologs and breast cancer in humans. Such experimentation would be replete with trial and error analysis as there is no indication from the specification, that amplification of WIP1 homologs on different regions of the human genome, are associated with breast cancer. Given that the region the WIP gene resides in is known to be amplified in breast cancer, it is not clear whether the association is due to amplification of WIP1 or another gene in the amplified region. Thus given the broad claims in an art whose nature is identified as unpredictable, the unpredictability of that art, the large quantity of research required to define these unpredictable variables, the lack of guidance provided in the specification, balanced only against the high skill level in the art, it is the position of the examiner that it would require undue experimentation for one skilled in the art to perform the methods of the instant claims as written.

***Response to Arguments***

7. The response traverses the rejection. The response asserts that the plain language of claim 1 states that the recited "WIP1 gene" is present in breast tissue sample of a human, which limits the nucleotide sequence of the gene to that which is naturally present in human tissues. The response asserts that human WIP1 coding sequences are known in the art and do not greatly differ from SEQ ID NO: 1, providing BLAST alignments with several Genbank entries for human WIP1. The response asserts that the last three entries of the table at page 6 of the

response indicates that the WIP1 coding sequence present in three different human cDNA clones is highly conserved. This argument has been thoroughly reviewed but was not found persuasive as the BLAST analysis refers to sequences which have 99% or great sequence identity with SEQ ID NO: 1. However, the breadth of the claims is broader, encompassing homologs with less percent identity to SEQ ID NO: 1. Accordingly, the scope of the claims does not bear a reasonable correlation to the scope of enablement provided by the specification. The rejection is maintained.

***Claim Rejections - 35 USC § 102***

8. Claims 1, 3, 57, 61, and 62 are rejected under 35 U.S.C. 102(b) as being anticipated by Kallioniemi et al. (herein referred to as Kallioniemi, *Proc. Natl. Acad. Sci. USA*, vol. 91, pages 2156-2160, 03/1994), as defined by Wu et al. (herein referred to as Wu, *Cancer Res.*, vol. 61, pages 4951-4955, 07/2001) and Genbank Accession number NM_003620 (1999, as set forth at pages 36-37 of the instant specification).

Wu teaches that the human WIP1 gene is located in the 17q22-23 region of chromosome 17 (see Figure 1 of Wu).

Kallioniemi teach a method of detecting and measuring DNA sequence copy number increases for the 17q22-24 region in several human primary breast tumors and breast cancer cell lines (instant claim 1; see Tables 1 and 2, page 2156, all of paragraph 5, and page 2157, all of paragraphs 1 and 2). Kallioniemi teach that copy number increases of the 17q22-24 region were found in 18% of primary breast tumors and 67% of breast cancer cell lines examined (see Tables 1 and 2 and page 2159, paragraph 2, lines 5 and 6 of Kallioniemi). This above method taught by

Kallioniemi involves comparative genomic hybridization in which the relative intensity of a fluorescent signal from a test chromosome (from tumor cells for example) hybridized with a labeled probe is compared to the intensity of a fluorescent signal from a control chromosome hybridized with the same probe that emits a different fluorescent color (instant claims 1, 57, 61, and 62; see page 2156, paragraph 2, lines 3-8 of Kallioniemi). Kallioniemi teaches that the probe/chromosome hybridizations of the above method were analyzed using a digital image analysis system that was based on either a Nikon SA or Zeiss Axioplan microscope equipped with a cooled charge-coupled device camera and a filter system consisting of a triple-band-pass beam splitter and emission filters and therefore the data was stored in an electronic video format (instant claim 3; see Figure 1 and page 2157, paragraph 3, lines 1-6 of Kallioniemi). Kallioniemi further teaches that three-color images derived from the above method were processed with a Sun IPX workstation using Scil-Image software for pseudocolor display and therefore the data was analyzed via video display and compared and compiled at a location where the data was transmitted (instant claim 3; page 2157, paragraph 3, lines 11-14 of Kallioniemi).

Although Kallioniemi does not teach the sequence which is amplified, nor does Wu teach the specific sequence detected by Kallioniemi, as stated in the MPEP in chapter 2100:

> Where the claimed and prior art products are identical or substantially identical in structure or composition, or are produced by identical or substantially identical processes, a prima facie case of either anticipation or obviousness has been established. In re Best, 562 F.2d 1252, 1255, 195 USPQ 430, 433 (CCPA 1977). "When the PTO shows a sound basis for believing that the products of the applicant and the prior art are the same, the applicant has the burden of showing that they are not." In re Spada, 911 F.2d 705, 709, 15 USPQ2d 1655, 1658 (Fed. Cir. 1990).

In the instant case, Kallioniemi teaches detecting amplification of 17q22-24 in a number of different tumors and cell lines. Thus, the office has sound basis for believing that some breast tumor samples which showed amplification of 17q22-q24 contained the gene which encoded the claimed sequences.

Claims 1, 3, 57, 59, 61, and 62 are rejected under 35 U.S.C. 102(b) as being anticipated by Orsetti (Orsetti et al; Oncogene, vol. 18, pages 6262-6270, 1999), as defined by Wu et al. (herein referred to as Wu, *Cancer Res.*, vol. 61, pages 4951-4955, 07/2001) and Genbank Accession number NM_003620 (1999, as set forth at pages 36-37 of the instant specification).

Wu teaches that the human WIP1 gene is located in the 17q22-23 region of chromosome 17 (see Figure 1 of Wu).

Orsetti teaches a method of detecting and measuring DNA sequence copy number increases over the entire 17q21-q24, including 17q21-qter, in 15 human breast tumors, and 3 of the entire long arm (see Fig. 2, Fig 5, and para bridging cols 1 and 2 of page 6264). The method taught by Orsetti includes PCR, FISH, and CGH (see page 6269). Orsetti teaches microscopy and digital image analysis (page 6269, fig 4; claim 3).

Although Orsetti does not teach the specific nucleotide sequence which is amplified, as stated in the MPEP in chapter 2100:

> Where the claimed and prior art products are identical or substantially identical in structure or composition, or are produced by identical or substantially identical processes, a prima facie case of either anticipation or obviousness has been established. In re Best, 562 F.2d 1252, 1255, 195 USPQ 430, 433 (CCPA 1977). "When the PTO shows a sound basis for believing that the products of the applicant and the prior art are the same, the applicant has the burden of showing that they are not." In re Spada, 911 F.2d 705, 709, 15 USPQ2d 1655, 1658 (Fed. Cir. 1990).

In the instant case, Orsetti teaches detecting amplification of 17q21-24 and 17q21-qter in a number of different breast tumors and cell lines. Thus, the office has sound basis for believing that some breast tumor samples which showed amplification of 17q21-q24 and 17q21-qter contained the gene which encoded the claimed sequences.

***Response to Arguments***

9. The response traverses the rejections under 35 USC 102 (b) and asserts that the claims have been amended to recite "comparing the test gene copy number to data for a WIP1 control gene copy number to detect WIP1 gene amplification" and "identifying the breast tissue sample as cancerous if there is amplification of the WIP1 gene in the breast tissue sample". This argument has been thoroughly reviewed but was not found persuasive as these limitations are inherent in the teachings of Kallioniemi and Orsetti. As set forth in the rejection above, Kallioniemi and Orsetti teach detecting amplification of 17q21-24 in a number of different breast tumors using CGH and CGH & FISH, respectively, to provide a test copy number and comparison to a control copy number. Although neither Kallioniemi nor Orsetti appreciate that WIP1 is in the region found to be amplified, this is a property of the region as evidenced by Wu. The identifying step does not distinguish the claims over the prior art because the fact that applicants have recognized an inherent result of a process taught in the prior art does not change how the process is performed. Merely recognizing something in an old process that was not known before is insufficient to render that process patentable. See MPEP 2112 I and II. The rejections are maintained.

***Claim Rejections - 35 USC § 103***

10. Claim 58 is rejected under 35 USC 102(a) as being unpatentable over Orsetti in view of Backman (Backman et al; US Patent 5,516,663).

Orsetti teaches a method of detecting and measuring DNA sequence copy number increases over the entire 17q21-q24, including 17q21-qter, in 15 human breast tumors, and 3 of

the entire long arm (see Fig. 2, Fig 5, and para bridging cols 1 and 2 of page 6264). The method taught by Orsetti includes PCR (page 6269). Orsetti does not teach amplification using LCR, however, Backman teaches amplification of targets using LCR (see col. 2). Additionally, it was known in the art at the time the invention was made that LCR was a suitable method of amplification (acknowledged in the specification, page 43). Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the invention was made to use LCR in the method of Orsetti in view of the teachings of Backman. The ordinary artisan would have been motivated to use LCR in the method of Orsetti because it was known in the art at the time the invention was made that LCR was an equivalent method of amplification for target detection.

11. Claim 63 is rejected under 35 U.S.C. 103(a) as being unpatentable over Kallioniemi or Orsetti, each in view of Pinkel (Pinkel et al; Nature Genetics, vol. 20, pages 207-211, 1998).

Kallioniemi teach a method of detecting and measuring DNA sequence copy number increases for the 17q22-24 region in several human primary breast tumors and breast cancer cell lines (instant claims 1 and 2; see Tables 1 and 2, page 2156, all of paragraph 5, and page 2157, all of paragraphs 1 and 2). Kallioniemi teach that copy number increases of the 17q22-24 region were found in 18% of primary breast tumors and 67% of breast cancer cell lines examined (see Tables 1 and 2 and page 2159, paragraph 2, lines 5 and 6 of Kallioniemi). This above method taught by Kallioniemi involves comparative genomic hybridization in which the relative intensity of a fluorescent signal from a test chromosome (from tumor cells for example) hybridized with a labeled probe is compared to the intensity of a fluorescent signal from a

control chromosome hybridized with the same probe that emits a different fluorescent color (instant claims 1, 54, 56, 57, 61, and 62; see page 2156, paragraph 2, lines 3-8 of Kallioniemi).

Orsetti teaches a method of detecting and measuring DNA sequence copy number increases over the entire 17q21-q24, including 17q21-qter, in 15 human breast tumors, and 3 of the entire long arm (see Fig. 2, Fig 5, and para bridging cols 1 and 2 of page 6264). This above method taught by Orsetti uses CGH (see page 6269).

Neither Kallioniemi nor Orsetti teach using microarray based CGH, however Pinkel teaches that arrays allow for high resolution analysis of DNA copy number variation using CGH. Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to improve the method of Kallioniemi or Orsetti with the microarray based CGH method of Pinkel. The ordinary artisan would have been motivated to improve the CGH methods of Kallioniemi or Orsetti with the microarray based method of Pinkel for higher resolution analysis of DNA copy number variations.

***Response to Arguments***

12. The response traverses the rejections and asserts that neither Pinkel nor Backman cure the deficiencies of Kallioniemi or Orsetti. This argument has been thoroughly reviewed but was not found persuasive for the reasons already made of record.

***Conclusion***

13. **THIS ACTION IS MADE FINAL.** Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO

MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

14. No claims are allowed.

15. Any inquiry concerning this communication or earlier communications from the examiner should be directed to examiner Jehanne Sitton whose telephone number is (571) 272-0752. The examiner can normally be reached Monday, Wednesday and Thursday from 9:00 AM to 3:00 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla, can be reached on (571) 272-0735. The fax phone number for this Group is (571) 273-8300.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

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For all other customer support, please call the USPTO Call Center (UCC) at 800-786-9199.

/Jehanne Sitton/
Primary Examiner
Art Unit 1634